

Loria and Stanley Webb of the Medical College of Virginia at Richmond, and Sidney Kibrick and Gordon Madge at Boston University. They reported their findings at a meeting of the American Federation of Clinical Research in Boston this week.

Coxsackie B virus has been suspected of playing a role in diabetes. Loria and his colleagues have explored its role in mice predisposed to acute onset juvenile diabetes. Specifically, they used the virus to infect mice having a genetic predisposition toward diabetes, as well as to infect mice that did not. After infection, the genetically predisposed animals came down with diabetes. The virus could also be seen attacking their pancreases. The

pancreas is the gland that makes insulin. In contrast, the nongenetically predisposed mice did not come down with diabetes.

"We are trying to see if this is, first of all, the only cause [of acute onset juvenile diabetes]," Loria told SCIENCE NEWS. "There may be many more causes. We'd like to know how the virus triggers the disease. Then we might think about how we can intervene."

One possible approach the investigators have suggested would be the manufacture of a coxsackie B vaccine. The vaccine could then be used to immunize children at special risk from acute onset juvenile diabetes. □

## T-mycoplasmas and male infertility

During the past several years it has become apparent that a bizarre microorganism, a cross between a bacterium and a virus, may be responsible for certain cases of male infertility. In one study, for instance, Hakan Gnarpe and Jan Fridberg of the University of Uppsala isolated the microorganism, called the T-mycoplasma, from the semen of men with unexplained infertility. They suspected that the organism might have triggered the problem and gave the men antibiotics. Afterward the wives of 30 percent of the men became pregnant (SN: 9/22/73, p. 182).

Now two studies reported in the December FERTILITY AND STERILITY provide further evidence that T-mycoplasmas can trigger certain cases of reproductive failure among men. Research conducted by the investigators may also lead to a highly effective means of treating T-mycoplasma-caused infertility, as well as offer new approaches to birth control.

In the first study, Dana M. Fowlkes, Gerald B. Doohar and William M. O'Leary at Cornell University Medical College took samples of ejaculated sperm from infertile men and cultured them for the presence of T-mycoplasmas. Those ejaculates found to contain the microorganism were put in one group. Those

found not to contain the microorganism were put in another group. The ejaculates from each group were then examined under the scanning electron microscope to see whether T-mycoplasmas actually adhered to the sperm in the ejaculates.

The sperm from the ejaculates containing the T-mycoplasmas could be seen to be coated with numerous spherical T-mycoplasmas, interlaced with fibrils, imparting a rough texture to the sperm.



A sperm without T-mycoplasmas (top). A sperm coated with T-mycoplasmas (bottom). □

The sperm had coiled tails, which may or may not be related to T-mycoplasma infection. In contrast, the sperm from ejaculates without the T-mycoplasmas were smooth. There was no evidence of T-mycoplasmas on them.

In the second study, Fowlkes, O'Leary and another Cornell scientist, John MacLeod, found that sperm from infertile men containing the T-mycoplasmas couldn't move as fast as sperm from infertile men without the microorganism. So they concluded that T-mycoplasmas may trigger infertility by decreasing the motility of sperm up the female vaginal tract. What's more, the binding of T-mycoplasmas may also allow them to slip past the normal microbial barrier of the female cervix.

Since their studies were submitted to FERTILITY AND STERILITY, the Cornell microbiologists and anatomists have also found that T-mycoplasmas don't have to be present to inhibit fertilization. Rather, they secrete a chemical that does it. "We are now working on isolating that material," O'Leary told SCIENCE NEWS. "Once we get that, then we can find out the actual biochemical lesion that causes all this to happen." Isolation of the chemical, he says, may also lead to a sure-fire therapy for T-mycoplasma-induced infertility. What's more, it may also offer a new approach to birth control. □

## WMO: Limited SST fleet won't hurt

The World Meteorological Organization (WMO) released a study in Geneva last week predicting that the number of supersonic transports (SST's) now planned for commercial operation would not hurt earth's ozone layer, as many had feared. The latest in a long series of often conflicting reports on the subject was released just as the U.S. Secretary of Transportation, William T. Coleman Jr., was holding hearings on whether the British-French SST, the Concorde, should be forbidden to land in American airports because of its noise.

The WMO announcement calls for international agreement on the total level of permissible emissions, but says that with the limited fleets now planned—a total of 30 to 50 planes—no "significant" effect on the ozone layer should occur. Indeed, it said any effect would likely be indistinguishable from "natural variation."

However, according to the study, some damage might be done if much larger fleets were eventually put into service, or if the SST's were designed to fly higher than the present 10-mile altitude. The ozone layer lies mainly between 12 and 15 miles above the earth, and severe depletion would cause greater amounts of ultraviolet light to penetrate, probably leading to increased incidence of skin cancer. □